

A Case of Cortical Vein Thrombosis With the Cord Sign

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Background: Computed tomography is performed in suspicious cases of cerebral venous thrombosis. Although an empty delta sign is not uncommonly reported, a cord sign is rarely reported. But this is, if present, pathognomonic, especially in the case of cortical vein thrombosis.

Case Description: A young man with antithrombin III deficiency sought emergency treatment for headache and seizure. Noncontrast computed tomography showed a typical cord sign in multiple sections. It seemed to be inde-

pendent from the adjacent pathologic findings in most sections. Brain magnetic resonance imaging verified it as a thrombosed vein in conjunction with acute venous infarction.

Conclusion: The cord sign should be considered for early and accurate diagnosis of cortical vein thrombosis.

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CEREBRAL VENOUS thrombosis (CVT) remains a challenging problem.¹ Invasive angiography is often needed. Newer diagnostic technology, such as magnetic resonance imaging (MRI), MR angiography (MRA), and MR venography (MRV), enables us to noninvasively investigate the cranial venous system.

Cerebral venous thrombosis may result from numerous conditions.¹ In many cases, the disease course is often unpredictable, mainly because of the seriousness and intricacy of the underlying causes. It should be diagnosed as soon as possible. Computed tomography (CT) is the method of choice for the early diagnosis of CVT.

Certain pathognomonic CT findings are indicative of CVT.²⁻⁴ An empty delta sign is a filling defect in the superior sagittal sinus. A visualized cortical vein, or the cord sign, is a useful indicator, especially in cortical vein thrombosis (CoVT). Because this sign is rarely reported, suspected cases should be scrutinized thoroughly; it is detectable even on noncontrast CT. Contrast CT is potentially harmful to the examinee. In the literature, a handful of CoVT cases and only a few cases with the cord sign have been reported.²⁻⁴ Moreover, the MRI-detected cord sign has been described in only one report.⁵ Here, we report a case of

CoVT caused by antithrombin III deficiency. Noncontrast CT and MRI show the cord sign and venous infarction in the paracentral gyrus.

REPORT OF A CASE

A 26-year-old man visited the emergency department in July 2000 because of a seizure. He had a 5-day history of throbbing headache, most severe in the temporal areas. History of febrile illness or head trauma was denied. At the emergency department, another seizure occurred, with preceding clonic movement of the right upper arm and secondary generalization. It was controlled with intravenous clonazepam.

He had a pulmonary embolism in July 1997. Laboratory investigation had found a decreased level of antithrombin III. After excluding other causes, physicians diagnosed him as having antithrombin III deficiency and prescribed warfarin. The patient stopped the anticoagulation treatment at will and had intermittently taken aspirin since 1998. His family history was unremarkable.

He was alert and well oriented on neurologic examination after seizure control. Results of cranial nerve and motor/sensory examinations were normal, without pathologic reflex. Noncontrast CT was

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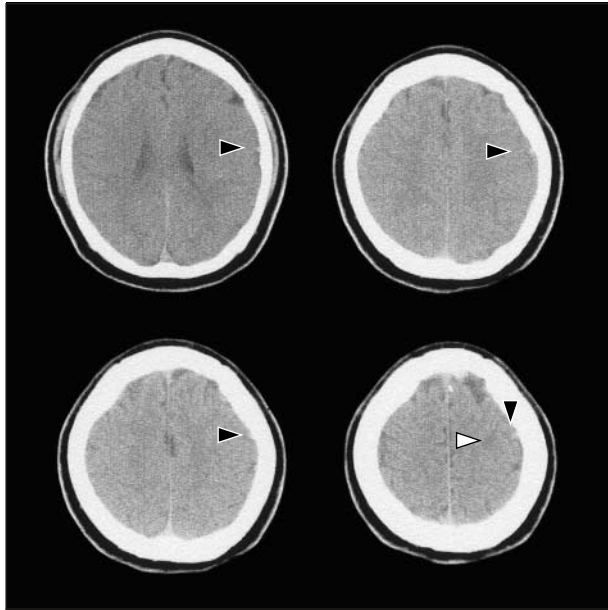


Figure 1. Noncontrast computed tomography shows the cord sign (black arrowheads). It is present at multiple continuous sections. A small low attenuated lesion (white arrowhead) is observable.

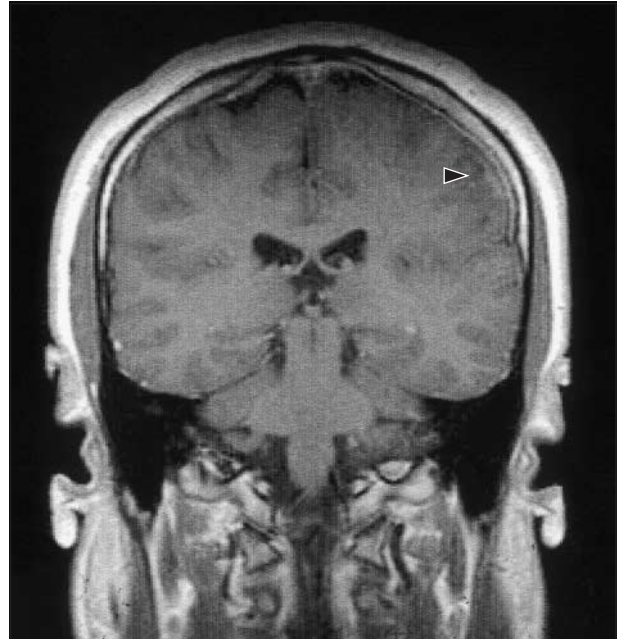


Figure 3. Postcontrast T2-weighted imaging shows a vertically elongated lesion on the surface of left hemisphere (arrowhead).

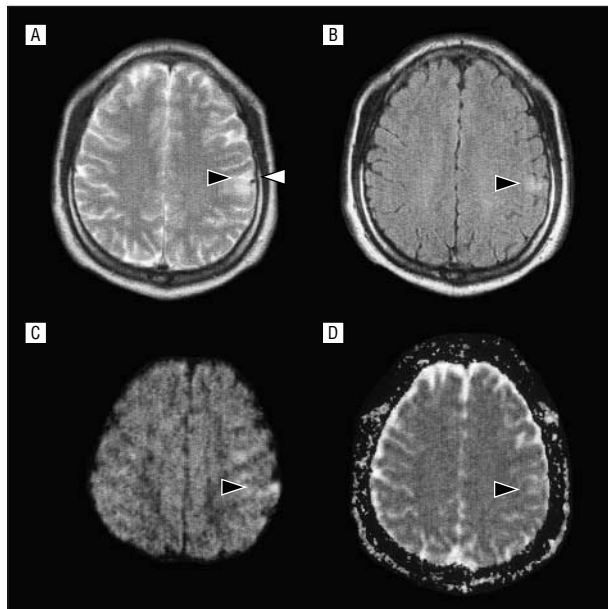


Figure 2. High signal intensity lesion is shown in the left paracentral gyrus (black arrowheads). A, T2-weighted imaging; B, fluid-attenuated inversion recovery imaging; C, diffusion weighted imaging; D, apparent diffusion coefficient map imaging. The thrombosed cortical vein is also evident (white arrowhead).

performed in the emergency department (**Figure 1**). A small, highly attenuated cord-like lesion was detected over the surface of the cortex. It was visualized in multiple continuous CT sections. Magnetic resonance imaging was also performed (**Figure 2** and **Figure 3**). T2-weighted and fluid-attenuated inversion recovery imaging revealed an acute infarction in the left paracentral gyrus, hyperintense on diffusion-weighted imaging and apparent diffusion coefficient map imaging. A CoVT appeared as a T2 low signal intensity lesion along the cor-



Figure 4. Brain magnetic resonance venography detects partial loss of signal in the mid segment of the superior sagittal sinus (arrowheads).

tical surface. It was pronounced in postcontrast T1-weighted imaging as a vertically elongated lesion. Magnetic resonance angiography detected no significant stenoses. Magnetic resonance venography revealed a partial filling defect in the mid-segment of the SSS (**Figure 4**).

The serum level of antithrombin III was 35% below normal. Echocardiography revealed patent foramen ovale with a right-to-left shunt. Intracardiac thrombi were

not present. A lung perfusion scan showed a perfusion defect in the apical segment of the right upper lobe that had remained unchanged since July 1997. Results of MRV of the lower extremities were normal. Anticoagulation was done. His headache gradually subsided. He was discharged and followed up without any additional thrombotic event.

COMMENT

The cord sign is rarely reported in the literature. It was first described by Buonanno et al.⁴ They presented an enhanced cortical vein abutting a subdural hematoma. Later, it appeared as a white spot on noncontrast CT. Rao et al³ also described the cord sign in a case. It was located in the periphery of an infarction. Macchi et al⁵ reported CT and MRI findings of CoVT. Noncontrast CT revealed hyperintensity of the superficial cortical vein. It was accompanied by ischemic infarction in the adjacent parietal cortex. In contrast with the empty delta sign, the cord sign is detectable without contrast enhancement.

Recently, Jacobs et al² reviewed case reports on isolated CoVT and found only 9 cases of isolated CoVT, including 4 of their cases and 1 case with the cord sign. None of their cases showed the sign. Although some studies have reported cases with the cord sign, these so-called cord signs were actually thrombosed sinuses on noncontrast CT rather than CoVT.⁶⁻⁹ Isolated CoVT is an exceptional presentation of CVT, and the cord sign is found solely in this subgroup.

Rao et al³ underlined the important role of neighboring pathologic characteristics in disclosing the cord sign. A bony structure or adjoining brain parenchyma could obscure the thrombosed vein. Therefore, it would be more easily detected by displaced brain parenchyma (by subdural hematoma) or damaged neighboring tissue (infarction). However, in our patient, only a portion of the CoVT was visualized in the periphery of the infarction. The remaining portion was not contiguous to the infarction.

A freshly thrombosed vein displayed hypointensity on T2-weighted imaging within 48 hours of symptom onset.⁵ Recently, Chu et al¹⁰ described a thrombosed superficial sylvian vein in the chronic phase on MRI. It appeared as a T1 high signal intensity lesion. In our case, the thrombosed vein was a T2 low signal intensity lesion. It looked like a vertical cord on the gadolinium-enhanced image. Considering the partial loss of signal in the mid-segment of the superior sagittal sinus, we presume that CoVT occurred in the vein draining to the superior sagittal sinus. This coincides with venous infarction in the paracentral gyrus. It was hyperintense on diffusion-weighted imaging, with increased apparent diffusion coefficient, which is compatible with vasogenic edema.^{11,12}

We have demonstrated isolated CoVT, visualized on noncontrast CT as the cord sign, and verified it as CoVT by MRI. It should be scrutinized in suspected cases and, if found, proper treatment should be instituted. Anticoagulation is an effective treatment and less dangerous than it was in the past.¹³ Early and accurate diagnosis of CoVT in its isolated form is important and critical to prevent its evolution into the more complicated CVT.

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